EFFECTS OF CIGUATOXIN-1 ON ELECTRICAL ACTIVITY RECORDED INTRACELLULARLY FROM RAT TAIL ARTERY IN VITRO. *Memoirs of the Queensland Museum 34(3): 454. 1994*:– Ciguatoxin-1 (CTX-1) is a lipid soluble toxin from the benthic dinoflagellate, *Gambierdiscus toxicus*; it is responsible for the disease ciguatera which exhibits a range of symptoms involving the peripheral nervous system. CTX-1 has been credited with a selective action on tetrodotoxin (TTX)- sensitive Na^+ channels and induces spontaneous nerve action potentials due to opening of sodium channels at normal resting potential. In this study the effects of CTX-1 on the rat tail artery have been investigated. Intracellular recordings were made from isolated sections of rat tail artery. Application of 0.002–0.2nM CTX-1 increased the occurrence of spontaneous excitatory junction potentials (SEJPs) and duration of the evoked excitatory junction potential (EJP), the decay phase no longer being fitted by a single exponential function. At 0.2nM CTX-1 also produced a large (25-30mV) maintained depolarization. The effects of CTX-1 were abolished by tetrodotoxin (0.3μM) and were calcium dependent. In addition EJPs and SEJPs were blocked by the purinoceptor antagonist suramin (1mM) and the maintained depolarization was blocked by the -adrenoceptor antagonist phentolamine (1μM). These data suggest the actions of CTX-1 are due solely to activation of the sympathetic nerves innervating the rat tail artery.

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RESPONSES OF VERTEBRATE NERVES TO CIGUATOXIN. *Memoirs of the Queensland Museum 34(3): 454. 1994*:– Electrophysiological studies were performed on a variety of nerve preparations from both mammals (rats and humans) and fish. Responses of human peripheral nerves to ingested ciguatoxin were assessed in the Sural nerves of 15 victims of ciguatera. In rats responses of the ventral coccygeal nerve in anaesthetised animals were studied after intoxication was induced by intraperitoneal injection of sub-lethal duses of ciguatoxin. In fish, isolated segments of the spinal nerves and the lateral line branch of the Vagus nerve of both 'carriers' and 'non carriers' of ciguatoxin were exposed to solutions of ciguatoxin in fish Ringer.

In all nerve preparations there were significant changes in a range of nerve conduction parameters including conduction velocity, amplitude, and the duration of refractory periods and the supernormal period. In all preparations there was a significant prolongation of an increase in the magnitude of the supernormal period. These changes conform with studies on isolated cells that suggest a fundamental action of ciguatoxin on Na^+ gating mechanisms.

Both rat and fish preparations have been used to assess the efficacy of a range of potential antagonists of the ciguatoxin response. In rats, ciguatoxin-induced changes in supernormality are unaffected by mannitol but significantly antagonised by lignocaine. In fish, lignocaine and tetrodotoxin antagonise the responses induced by ciguatoxin.

It has also been established that the nerves of fish respond to ciguatoxin in a similar manner to those of mammals and it is suggested that fish may have evolved some degree of protection against ciguatoxin by mechanisms that do not involve the Na^+ channel.

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